with pain, vomiting and even jaundice and fever. Sometimes it is extremely difficult to differentiate these temporary hyperdistensions from the mechanical effect of the passage of a stone. That this can occur has been proved at secondary explorations.

The great variance between recurrences after radical surgery of the gall-bladder and those that take place after conservative surgery leads to the manifest conclusion that radical treatment gives the greater prospect of a permanent cure. For the present, however, radical surgery, although it may fall short of being ideal surgery, is the best means one has of removing a pathological condition and its pernicious effects. In general most authorities agree with this viewpoint. Gall-bladders should always be removed at the time of the primary operation, as their retention in the presence of persisting infection come to represent an infected focus from which the system is intermittently flooded with toxins. If in these cases secondary cholecystectomy is refused by the patient, chronic pancreatitis may develop in addition to the chronic cholecystitis and invalidism may develop for the remainder of the patient's life.

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THE ETIOLOGY OF PNEUMONIA.*

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When the physician has made a clinical diagnosis of pneumonia there are several questions which immediately present themselves for solution. Is it primary pneumonia or is the pneumonia secondary to some preëxisting pathologic condition? Is it lobar pneumonia or bronchial pneumonia? How many lobes are involved?

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Finally, and in some respects the most important question of all, Which of the various bacteria are responsible for the infection?

I am aware that many clinicians will disagree with me on the relative importance of the bacteriology of pneumonia. Strangely enough, these same clinicians will admit the necessity for bacteriologic information in other infectious diseases but deny its importance in pneumonia. We are accustomed to make early and thorough examinations for the infectious agent in meningitis; we are accustomed to bacteriologic examination in dysentery and other intestinal infections. Even in endocarditis, arthritis and certain other rarer infections, we are anxious to find the etiologic agent.

Why, then, have we been so long in coming to a realization of the importance of a bacteriologic diagnosis in pneumonia? I think there are two reasons for this lack of interest: (1) Because the bacteriology of pneumonia has been one of the last branches of this subject to be worked out accurately, and (2) because there has been no specific treatment for pneumonia. Neither of these reasons, however, hold good at the present time. The various groups of pneumococcus and streptococcus have been accurately defined. Furthermore, we have a specific treatment for pneumococcus type I pneumonia and there are good reasons for believing that an efficient specific therapy will soon be worked out for the other types. Then, too, the prognostic value of a bacteriologic diagnosis in pneumonia has not been sufficiently emphasized. In respect to prognosis the microörganism present has great significance: For example, Bacillus Friedländer pneumonia is nearly always fatal. The mortality rate in pneumococcus pneumonia varies greatly with the type. As worked out by Dochez and Gillespie at the Rockefeller Institute the rate for pneumococcus type III pneumonia may be as high as 50 per cent. On the other hand, pneumococcus type IV shows a rate of only 10 to 15 per cent. A similar divergence is found in streptococcus pneumonia. Streptococcus hemolyticus pneumonia has a death-rate varying from 40 to 60 per cent, whereas Streptococcus viridans pneumonia is one of the mildest forms encountered. In our Camp Upton series the death-rate for Streptococcus viridans pneumonia was under 10 per cent.

It would appear from these figures that in many instances the type of bacterium present had a more important bearing on the prognosis in pneumonia than either the character or the extent of the consolidation. Personally, if I must have pneumonia I am indifferent whether it shall be lobar pneumonia or bronchial pneumonia, nor am I particularly concerned whether I have two lobes or three lobes involved; but I do care very much whether my infection is caused by Friedländer's bacillus or pneumococcus type IV. Furthermore, if streptococcus is the causative agent I am very anxious to know whether it is Streptococcus hemolyticus or Streptococcus viridans. There is the difference here between 1 in 2 chances and 9 in 10 chances for recovery.

Bacteriology of Pneumonia. Practically speaking, there are only three bacteria that are commonly concerned in the etiology of pneumonia: Pneumococcus, streptococcus and Bacillus influenzæ. Friedländer's bacillus, Staphylococcus aureus, meningococcus and other microörganisms are occasionally responsible for this disease, but they constitute such a small percentage of the whole that they must be looked upon as rarities.

The pneumococcus is the usual cause of lobar pneumonia. Dochez and Gillespie have classified pneumococci on the basis of their biologic characteristics into four groups. Thus all strains of pneumococcus type I cross-agglutinate and cross-protect. The same with types II and III. Group IV is the heterogeneous or waste-basket group and is composed chiefly of unrelated strains which do not fall into the other three groups. The first three groups are known as the "fixed types" of pneumococcus. They are highly parasitic as a rule, and except for type III rarely occur in healthy mouths. The strains that compose group IV are less virulent and are frequently encountered in healthy mouths.

At the time the studies on pneumococcus types were carried out at the Rockefeller Institute the fixed types were responsible for about 80 per cent of all lobar pneumonias, the remaining 20 per cent being caused by the group IV strains. During the two big epidemics of influenza this proportion was considerably upset by the relative increase in group IV pneumonias, but it is interesting to note that during the winter 1920-1921, in 200 cases of pneumococcus pneumonia typed at Bellevue Hospital the distribution has been as follows:

Pneumococcus type I	86 cases	43.0 per cent.
Pneumococcus type II	39 "	19.5 "
Pneumococcus type III	34 "	17.0 "
Total	159 "	79.5 "
Pneumococcus group IV	41 "	20.5 "

It is clear from these figures that the disturbance in the incidence rate produced by the influenza epidemic was only temporary and that the incidence of the fixed types of pneumococcus pneumonia is the same now as it was before the epidemic of influenza occurred.

The streptococcus is the organism most frequently found in bronchopneumonia. Here, again, we must classify into the "hemolytic" and "viridans" groups. Streptococcus hemolyticus causes a severe form of lobular pneumonia very frequently complicated by multiple abscesses and empyema. Streptococcus viridans is often recovered from the small patches of terminal bronchopneumonia discovered at autopsy in cases of chronic systemic disease. Apart from this association with terminal bronchopneumonias, however, the Streptococcus viridans produces a very mild form of pneumonia, the mortality rate being less than 10 per cent. It is rarely complicated by empyema. It should be emphasized that both types of streptococcus pneumonia usually occur as a complication or sequel to some preceding disease or injury. During the war the hemolytic streptococcus attained such a high degree of virulence that it appeared in some instances to excite a primary pneumonia, but such cases were exceptions to the rule.

The influenza bacillus also produces a characteristic type of pneumonia, best exemplified by the nodular hemorrhagic pneumonias so frequently seen during the big epidemic. Unfortunately for the pathologist this organism rarely occurs in pure culture, so that it is often difficult to say just what part of the process is due to the influenza bacillus and what part to the other bacteria present. A study, however, of the occasional cases of pure Bacillus influenzæ pneumonia and comparative studies on experimental Bacillus influenzæ pneumonia in monkeys indicate that this microorganism is capable of setting up a bronchitis and bronchiolitis with peribronchial consolidation—in other words a true bronchopneumonia with hemorrhage and edema, sometimes succeeded in the later stages by emphysema and bronchiectasis.

The rarer forms of pneumonia will not be discussed at the present time. The Friedländer bacillus produces a typical lobar pneumonia usually fatal, fortunately rare. Staphylococcus aureus pneumonia is another unusual form, described by Chickering[1] as a purulent bronchopneumonia with abscesses. Like Bacillus Friedländer pneumonia it is usually fatal.

The Mode of Infection in Pneumonia. The pneumococcus, like most other bacteria, attacks the human host through the nose or mouth. The mere presence of virulent pneumococci in the pharynx is not sufficient, however, to excite a pneumococcus pneumonia. It is quite possible that the bacteria reach the trachea through the inspired air. Recent experiments[2] indicate that lobar pneumonia is bronchiogenic rather than hematogenous in orgin. The constant production of the disease in monkeys by intratracheal injection and the constant failure to produce it by subcutaneous or intravenous injection are believed to be conclusive evidence that infection in lobar pneumonia takes place not through the blood stream but by way of the respiratory passages. The hematogenous theory has apparently depended upon the clinical observation of cases in which the pneumococcus was shown to be present in the blood at the time of onset of clinical symptoms of pneumonia or in occasional instances before the clinical symptoms of pneumonia had appeared. That the interpretation of this observation as indicative of the hematogenous origin of pneumonia is incorrect, is established by the fact that in pneumonia experimentally produced by the intratracheal injection of pneumococcus the organisms have appeared in the blood stream within six to twenty-four hours after injection— frequently before clinical evidence of pneumonia or elevation of temperature have developed.

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[1] Jour. Am. Med. Assn., 1919, 72, 617.

[2] Blake, F. G., and Cecil, R. L.: Jour. Exper. Med., 1920, 31, 403, 499.

When a monkey is injected intratracheally with virulent pneumococci and killed a few hours later it can readily be demonstrated that in experimental pneumonia, at least, the initial invasion of the lung occurs somewhere comparatively close to the hilum. The earliest inflammatory changes are found here and microscopic sections taken through this region show pneumococci penetrating the walls of the large bronchi and invading the peribronchial and perivascular lymph spaces. Furthermore, sections from the same lungs taken nearer the periphery show no pneumococci in the lumina of the bronchioles or alveoli, but they may be present in large numbers in the alveolar walls. It is quite clear that invasion and spread of pneumococci in the lung in lobar pneumonia is by way of the perivascular, peribronchial and septal tissue and lymphatics and that the alveolar structure is primarily infected by spread of the pneumococci from the grosser framework of the lungs into the alveolar walls. With further advance of the process to the stage of hepatization, pneumococci pass out from the alveolar walls into the alveolar spaces together with the exudate. Although by no means an absolute rule, hepatization usually occurs first in the posterior and upper parts of the lower and in the posterior parts of the upper lobes. This distribution in no way precludes early extension of the consolidation as far as the pleura, since the pleura is comparatively near the large bronchi and vessels in the part of the lobe proximal to the hilum. As the disease progressses, hepatization spreads to the distal portions of the lobe until complete or nearly complete lobar consolidation has occurred.

Experiments with Streptococcus hemolyticus on monkeys[3] indicate that the mode of infection in streptococcus pneumonia is similar to that in pneumococcus pneumonia. The infection is bronchiogenic and primarily affects the interstitial framework of the lung. The reaction of the tissue to the streptococcus is different, and therefore the pathologic picture is different, but the mode of infection is apparently the same for the two infections.

Coming now to the influenza bacillus a different mechanism appears to be at work.[3] In Bacillus influenzæ pneumonia the infection travels down the bronchial tree into the bronchioles and subsequently involves the neighboring alveoli by contiguity. Influenza bacilli are present in large numbers in the bronchioles and to a less extent in the adjacent alveoli. They are rarely found in any part of the interstitial tissue, and as MacCallum has pointed out the lymphatics are noticeably free from involvement in Bacillus influenzæ pneumonia. This may explain why the influenza bacillus rarely invades the blood stream. The influenza bacillus lacks the invasive character possessed by the pneumococcus and streptococcus, consequently the lesions produced by this organism are local while those produced by pneumococcus and streptococcus involve extensive areas. The influenza bacillus, growing in a sense outside of the body, injures chiefly contiguous parts, probably by means of toxic substances which it secretes. The pneumococcus and streptococcus, invading the lymphatics, reach all parts of the lung and excite a massive lesion.

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[3] Blake, F. G., and Cecil, R. L.: Jour. Exper. Med., 1920, 32, 401.

[4] Ibid., 719.

The Epidemiology of Pneumonia. In the United States pneumonia is an endemic disease, though it also occurs in epidemic form. The United States census for 1900 shows that more than 10 per cent of all deaths are due to some variety of pneumonia, and statistics seem to indicate that the incidence is increasing. In view of the fact that so many healthy individuals are pneumococcus carriers it was formerly assumed that most cases of pneumonia were autogenous infections. During the past few years, however, important studies on the epidemiology of pneumonia[5] have been carried out at the Hospital of the Rockefeller Institute, the results of which may be briefly summarized here.

In a study of 398 normal individuals type I and type II pneumococcus were practically never encountered in the buccal secretions. Type III, on the contrary, was found in 11 per cent of the cases. Group IV was encountered most frequently, appearing in 27 per cent of normal salivas. In contrast to these figures the Rockefeller Institute investigators showed that in a series of 454 cases of lobar pneumonia either pneumococcus type I or type II was present in 62 per cent of the cases. Type III was present in only 13 per cent, group IV in only 20 per cent of the cases; in other words, the pneumococci most commonly found in the mouth secretions of normal individuals give rise to a minority of cases of lobar pneumonia. This appears to indicate that pneumonia due to types I and II does not arise from infection with the pneumococcus which is carried in the mouth, but that infection with these organisms occurs from without.

A study was also made of the types of pneumococcus present during convalescence in pneumonia. This study revealed the fact that only in exceptional instances does one find for a considerable time after recovery the same type of pneumococcus with which the individuals was infected during the disease. Usually the infecting organism was found to have disappeared in three or four weeks after the illness. After its disappearance either no pneumococcus is present or the infecting type has been supplanted by one of the types usually found in normal saliva. These observations afford still further evidence that infection in pneumonia occurs from without.

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[5] Stillman, E. G.: Jour. Exper. Med., 1916, **24**, 651; ibid., 1917, **26**, 513.

Pneumococcus Carriers. Carriers of virulent pneumococci probably play an important part in the dissemination of the disease. The Rockefeller Institute workers showed that a considerable percentage of persons intimately associated with patients suffering from pneumonia harbor in their mouths pneumococci of the same type as those in the patient. Whereas pneumococcus types I and II are almost never found in normal salivas, it was shown that in a large series of healthy individuals in contact with cases of lobar pneumonia 13 per cent were pneumococcus type I carriers and 12 per cent pneumococcus type II carriers. It was also shown that pneumococcus is more frequently present in the dust of rooms in which pneumonia has occurred than in those in which no pneumonia has existed. Moreover the type of pneumococcus found in the dust corresponds to the type of pneumococcus found in the patient.

All that has been said in regard to the epidemiology of pneumococcus pneumonia probably applies with equal force to Streptococcus hemolyticus pneumonia. Although the hemolytic streptococcus is often found in normal tonsils, it is reasonable to suppose that it exists there in a comparatively harmless form. The epidemics of streptococcus pneumonia are probably caused by a few strains of virulent streptococcus which are passed on from one case to another by carriers or by actual contact. The contagious character of erysipelas, tonsillitis, puerperal fever and scarlet fever (probably a streptococcus infection) all support the idea that in streptococcus pneumonia as it existed for example in the army camps, the disease is transmitted from man to man by direct or indirect contact.

Methods of Making a Bacteriologic Diagnosis in Pneumonia. The bacteriologic examination of the sputum can be accurately made in many cases by direct plating of the material on blood agar. This method, however, is slow and for that reason undesirable. The mouse method is undoubtedly the quickest and at the same time the most reliable method of determining the type of organism in pneumonic sputum. By this method a positive diagnosis can usually be made in twelve to eighteen hours. 0.5 cc of washed sputum emulsified in sterile normal salt solution is injected intraperitoneally into a white mouse. If pneumococcus is present an agglutination test can be performed at once on the peritoneal exudate and the type determined. If the infectious agent be streptococcus or Bacillus influenzæ, or even Friedländer's bacillus, the mouse exudate will indicate it, for the peritoneum of the mouse is an ideal culture medium for all of these bacteria.

The Krumwiede method of examining the sputum is quick and reliable, but gives information only concerning the three fixed types of pneumococcus.

The Avery method is accurate when one uses very clean sputum in which the pneumococcus is present in practically pure culture. Unfortunately specimens of this character are not often obtained.

When sputum is not obtainable the diagnosis of pneumococcus types I, II and III pneumonia can sometimes be made very promptly by a precipitin test on the urine. Positive reactions, however, are usually obtained on severe cases only.

Other and less frequently available methods of making the diagnosis are by blood culture, lung puncture, examination of pleural and spinal fluid, etc. Examination of the sputum, however, remains the method of choice.

General Remarks. An accurate bacteriologic examination of the sputum is necessary before serum treatment can be rationally applied. If pneumococcus is demonstrated the type must be determined. If pneumococcus type I is present the administration of type I serum is to be seriously considered. The value of serum treatment has not yet been demonstrated for the other types.

In case Streptococcus hemolyticus is present the physician must watch the circulation and be on guard for abscess and empyema. On the other hand if nothing but Streptococcus viridans is present in the sputum, the prognosis is excellent. The influenza bacillus will only rarely be found in pure culture, but its presence in large numbers should be considered significant. In such cases there is danger of slow resolution and chronic bronchitis with occasionally emphysema and bronchiectasis.

In cases in which resolution is delayed the sputum should always be searched for tubercle bacilli. In cases of relapse a complete retyping is indicated, as relapses are often infections by a different microörganism.

The time has come when medical men should think of pneumonia in terms of the causative agent rather than in terms of anatomy or pathology—important as the latter aspects of the disease are. In consideration of the facts above presented, it would seem that the character of the infectious agent in pneumonia were more significant than the location or the type of consolidation. It is difficult at best to make accurate distinctions between lobar and lobular pneumonia, or to say in every case just how many lobes are involved in the process. On the other hand we now have at our disposal accurate and rapid methods for the determination of the infecting organism in pneumonia, and these methods are available to all practitioners, certainly to all practitioners in the larger cities. Would it not therefore be a step in advance to speak less of lobar and bronchial pneumonia and more of pneumococcus, streptococcus and Bacillus influenzæ pneumonia? The writer believes that progress in the specific treatment of pneumonia is in large measure dependent upon the adoption of such a point of view.